ANOMALOUS SYMPTOMS FOLLOWING TRAUMATIC HEMORRHAGE INTO THE SPINAL CORD.

LOSS OF KNEE-JERK WITH ANKLE-CLONUS AND GLUTEUS-CLONUS.[1]

By William M. Leszynsky, M.D.,

CONSULTING NEUROLOGIST TO THE MANHATTAN EYE AND EAR HOSPITAL; NEUROLOGIST TO THE DEMILT DISPENSARY, NEW YORK CITY, ETC.

Charles N., 35 years of age, born in Sweden, single, carpenter, was first seen by me May 26, 1898. Soon after, he disappeared from observation, but returned January 21, 1899, and gave the following history:

He was in perfect health until June 2, 1896. At that time he fell from the second floor to the basement in an unfinished building and struck on his buttocks. He was unable to get up, as he immediately became paralyzed in both lower extremities, necessitating his removal to the Presbyterian Hospital, where, six hours after the accident, the following condition was noted: "Loss of power in both lower extremities. Sensation lost in the legs to about three inches above the knees; normal in thighs. Areas in leg where tactile sense is preserved, but pain sense is lost. There is an area of anesthesia on right buttock, crescent-shaped, extending about 2½ inches from intergluteal fold. Retention of urine. Constipation." A few days later there was loss of control over the anal sphincter. The patient remained in the surgical ward and gradually improved. He was transferred to the medical division November 16, 1896, where the record shows that there were "paraplegia and some cystitis. Both knee-jerks were diminished and marked ankle-clonus was on both sides." After a sojourn of nine months he was discharged from the hospital, March 10, 1897, nearly two years ago.

He now complains of frequent and almost constant numbness, of a burning sensation in both feet and legs, and of weakness in both lower extremities. He can only walk with the aid of two crutches. He has frequent incontinence of urine, and exaggerated bladder reflex. His bowels are regular and he has good control of the sphincter. Sexual power is not decreased, and he has never suffered from priapism. He sleeps well, and his appetite and digestion are good. No previous history of alcoholism or syphilis.

Status Praesens.—He is a well developed and healthy looking man. The heart, lungs and upper extremities are normal. With slight assistance he is able to stand for a few moments.

---

[1] The patient and report were presented before the New York Neurological Society, February 7, 1899.

While doing so there is considerable hyper-extension at left knee-joint. In his effort to stand, marked clonus and trepidation of both lower extremities occur. There is incomplete paraplegia with slight spasticity, affecting the adductors of both thighs and the calf muscles. Evidence of local atrophy is absent, but there is general diminution in the consistency and bulk of all muscles, equally distributed in both limbs, as compared with the highly developed and muscular upper extremities.

Measurement.—Circumference of lower extremities, 6½ inches above patella, R. 18 inches, L. 18 inches; 6½ inches below patella, R. 12¾ inches, L. 12¾ inches.

In the sitting position he is able to fully extend both legs. In recumbency he can elevate the right limb to its full height, while the left can only be raised about three-fourths of the same distance. Both feet are in a condition of talipes equinus, but there is neither ankylosis nor contracture. In the right foot there is only slight power in dorsal flexion. In the left there is none. While lying in the prone position voluntary flexion of legs upon thighs is impossible. There is no evidence of deformity of vertebral column. Muscular resistance to passive movement is quite feeble in the posterior thigh muscles, the crural group and the ilio-psoas on both sides. The power of abduction (gluteus medius and gluteus minimus) is only slightly impaired, while the outward rotation of both thighs (gluteus maximus) is feeble. All of the paretic muscles are flaccid.

Electrical Examination.—Shows quantitative decrease in faradic irritability. Anterior crural nerve and muscles, R. 32 mm., L. 52 mm.; peroneal nerve, R. 25 mm., L. 25 mm.; tibial group of muscles, R. 50 mm., L. 55 mm.; biceps, R. 50 mm., L. 50 mm.; gluteus maximus, R. 50 mm., L. 50 mm. All contractions are slow. The reaction to the galvanic current presents nothing abnormal.

Both plantar reflexes are active. The Achilles-reflex is highly exaggerated on both sides. Ankle-clonus, which is quickly elicited in the usual manner, is equally well marked and persistent on both sides. It is also readily produced by percussion over the Achilles-tendon, with or without dorsal flexion of the foot, and while the patient is kneeling. In fact, the irritability of the gastrocnemius muscle itself is so exalted that a gentle tap upon it will at times produce the same degree of clonus, especially on the left side. This is not influenced in its activity by forced flexion of the great toe.[2] The left knee-jerk is absent. The right knee-jerk is hardly perceptible, if present at all, with the strongest reinforcement. Percussion just above or below the patellar tendon on either side produces slight contraction of the vastus externus only, but no knee-jerk (except as above mentioned). The contraction of this muscle alone is manifestly too weak to produce extension of the leg, and thus verify the claim of Sherrington[3] that the vastus externus is the chief muscle involved in the production of the knee-jerk. Sternberg has also demonstrated that there is no necessary relationship between increase of tendon reflexes and increased muscular tonus, and this latter conclusion would seem to apply in this case, as the mechanical irritability of the vastus externus is increased while the knee-jerk is absent.

---

[2] From a series of experiments on dogs and rabbits Sternberg has shown (Sitzungs-bericht der Kais. Akademie der Wissensch in Wien, 1891, Vol. C., Part 3) that on percussion over the Achilles-tendon the contraction is not limited to the gastrocnemius, but also includes the soleus and plantaris muscles.

From the fact that voluntary extension of the leg upon the thigh is possible, it would seem that the reflex arc is interrupted in its sensory portion within the cord. It is an open question in this particular case as to whether the contraction of the vastus externus is purely reflex or of an idiomuscular type.

Sudden and forcible pressure downward, or percussion below the origin of the gluteus medius muscle on both sides, produces a distinct and persistent clonus which is difficult to suppress, but ceases when the patient forcibly flexes the trunk upon the thighs. The cremasteric reflex is present, but feeble, on both sides. The abdominal reflex is present on the right and absent on the left side. The anal reflex is preserved.

Examination of the urine indicates the presence of a mild degree of cystitis. (Sp. gr., 1029; acid. Excess of phosphates. Muco-pus.)

The only objective disturbance of cutaneous sensibility is found almost symmetrically situated in both lower extremities, as outlined in the accompanying chart. In this area tactile sensibility is preserved, while there is complete analgesia and marked impairment of temperature sense. A very strong faradic current with the wire brush applied over the region produces a very slight sensation of pain. The muscular sense is normal in both lower extremities.

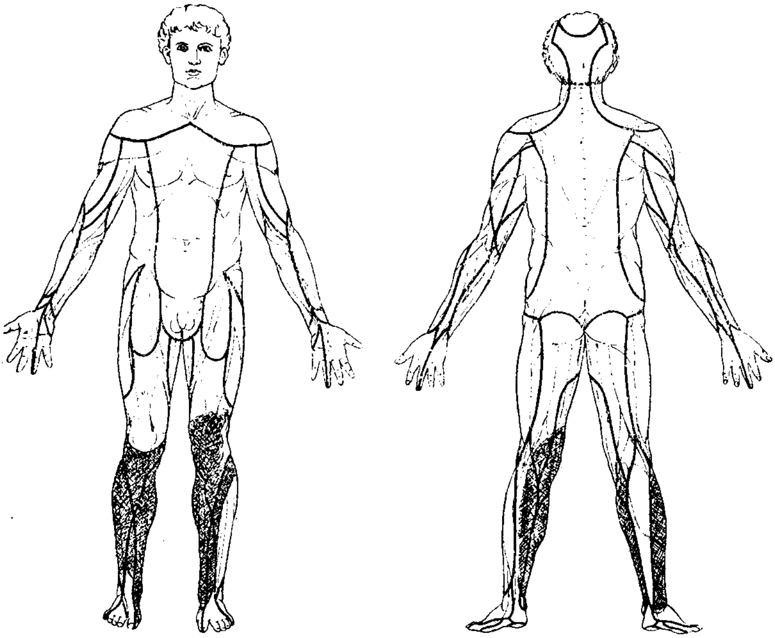
It will be observed that while this area of analgesia and thermoanesthesia does not accurately correspond with the picture of sensory disturbance resulting from a complete destruction of the third and fifth lumbar segments, it is sufficiently well defined to enable us to say with some degree of certainty that the lesion producing it is located in these segments. The dissociation of sensibility, as manifested in the preservation of the tactile sense, and its bilateral and symmetrical distribution, substantiates the conclusion as to its spinal origin.

---

[3] Brit. Med. J., 1892, Vol. I., p. 654.

It is also interesting to note that the area of anesthesia corresponds to the anatomical peripheral distribution of the sciatic (peroneal) and anterior crural (long saphenous) nerves, which innervate respectively the muscles paralyzed in this case, i. e., the biceps and tibial group, and the crural group. From a study of the functions of the various spinal segments, it has been assumed that these muscles are represented in the second and fourth lumbar segments.

The only reflexes that are absent are the knee-jerk and the abdominal reflex on the left side, while both cremasteric reflexes are weak. This would indicate involvement of their spinal centers, situated between the two lowest dorsal and the fourth lumbar segments.



The shaded areas in the lower limbs indicate the regions of preserved tactile sensibility, complete analgesia, and marked impairment of temperature sense.

The most unusual feature in this case, however, is the absence of the knee-jerk and the presence of ankle-clonus. It seems that this condition occurs in spinal cord lesions limited to the lumbar segments containing the reflex center for the knee-jerk, which at the same time cut off or damage the so-called inhibitory fibers in the motor tract communicating with the lower reflex centers.

If we would accustom ourselves to look upon ankle-clonus as concurrent with an exaggerated Achilles-reflex, and analogous to patella-clonus, which is so frequently associated with a highly exaggerated knee-jerk, the anomalous condition of ankle-clonus with loss of knee-jerk would become more comprehensible.

From a study of 1900 cases observed in the psychiatric clinic at Jena, Ziehen[4] found that excessive increase of the Achilles-reflex without foot-clonus was exceedingly rare. Foot-clonus without distinct increase in the Achilles-reflex he observed only twice. All of the other reflexes in my case whose spinal centers are in the sacral portion of the cord are either active or exaggerated.

The pronounced and excessive clonus affecting the deeper gluteal muscles is a condition that is rarely observed, and probably indicates an irritative character of the cord lesion, as well as damage to the inhibitory fibers.

It is a common experience to note the loss or diminution of sexual power in traumatic lesions affecting the lower portion of the cord. The preservation of function in this case may be explained by the escape from damage of the genital centers in the sacral segments. From the foregoing, we may conclude that primarily, there was a traumatic hemorrhage into the lumbar cord, with irregular disintegration of structure in the upper lumbar segments. This has probably resulted in a secondary degeneration affecting symmetrically the motor tract below this area, and in the formation of a cavity in this region. The lesion evidently extends irregularly from about the tenth dorsal to the upper sacral segments.

---

[4] Ziehen, Deutsch. med. Woch., 1894, Nos. 33 and 34.

---

61. UEBER PRURITUS ALS SYMPTOM DER PROGRESSIVEN PARALYSE (Pruritus as a Symptom of General Paresis). A. Sarbo (Pester med. chirurg. Presse, 33, 1897.

In two cases the author observed a persistent and intense pruritus as an early symptom of general paresis. This symptom disappeared as the disease progressed.

62. BEITRAG ZUR ÆTIOLOGIE DER PROGRESSIVEN PARALYSE (Contribution to the Etiology of General Paresis). W. Eccard (Ver. Bl. der pfälz. Ærzte, 13, 1897, No. 11).

The author believes that without syphilis there can be no general paresis. In a study of 35 cases, 24 men and 11 women, in the men a positive history was obtained for 22, 91.9 per cent., in the women, 8 cases had been infected, 72 per cent.